CHAPTER 68
Acute Vertigo and Imbalance

KEY TEACHING POINTS

- Most patients with isolated acute dizziness have benign peripheral vestibular disease (e.g., vestibular neuronitis).
- The probability of stroke in patients with acute dizziness is low, only 3% to 4%. Most patients with dizziness from stroke have additional neurologic findings, such as dysarthria, ophthalmoparesis, visual field defect, or focal motor or sensory findings.
- Nonetheless, 5% to 17% of patients with dizziness from stroke have isolated acute dizziness. In patients with isolated dizziness, 3 neuro-ophthalmologic findings greatly *decrease* probability of stroke: the combined presence of an *abnormal* head impulse test, *no* direction-changing nystagmus, and *no* skew deviation.

I. INTRODUCTION

Acute sustained vertigo and imbalance, often associated with nausea and vomiting, is collectively called **acute vestibular syndrome** (or **acute vestibulopathy**). Most affected patients have benign disorders of the peripheral vestibular system, such as dysfunction of the vestibular nerve (vestibular neuritis) or labyrinth (labyrinthitis). A few affected patients, however, are experiencing serious strokes of the cerebellum or brainstem, problems that may rapidly cause coma and death from acute hydrocephalus or brainstem compression.[1-3]

The full syndrome of brainstem stroke causing vertigo is described in Chapter 62 (see lateral medullary, or Wallenberg, stroke) and the syndrome of cerebellar infarction is described in Chapter 65. Nonetheless, 5% to 17% of strokes causing dizziness present as *isolated* dizziness or vertigo without other telltale cerebellar and brainstem findings.[4,5] This chapter focuses on these patients and discusses additional bedside findings that help distinguish stroke from peripheral vestibular disease.

II. THE FINDINGS

The additional findings that suggest stroke in acutely dizzy patients are *normal* bilateral vestibuloocular reflexes (detected by the head impulse test), skew deviation, abnormal visual tracking (saccadic pursuit), and direction-changing nystagmus.

A. THE VESTIBULOOCULAR REFLEX

In healthy humans, any head movement is involuntarily matched by opposing conjugate movements of the eyes through the actions of the **vestibuloocular reflex**. Without this reflex, it would be impossible to focus on objects when walking, riding,

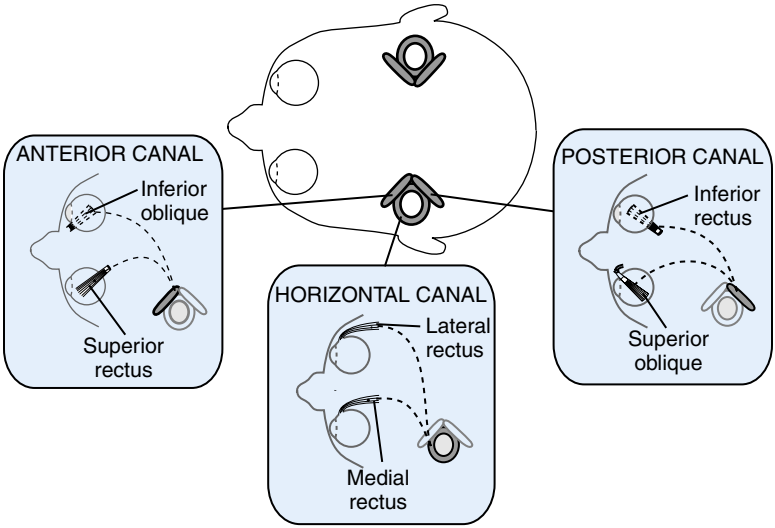

FIG. 68.1 CONNECTIONS BETWEEN SEMICIRCULAR CANALS AND EYE MUSCLES. Each of the blue-shaded boxes illustrates the orientation and specific connections between the semicircular canals—the anterior canal on the *left,* the horizontal canal in the *middle*, and the posterior canal on the *right*—and specific eye muscles (in these drawings, the semicircular canals are greatly magnified). Importantly, there are six semicircular canals (three on each side) and 12 eye muscles (six on each side). Therefore, each semicircular canal is yoked to two eye muscles, one on each side; these muscles pull the eyes in a conjugate manner *in the same plane* as the paired canal. The anterior canal is linked to the ipsilateral superior rectus and contralateral inferior oblique (both muscles are oriented in the same plane as the canal); the horizontal canal, to the ipsilateral medial rectus and contralateral lateral rectus; and the posterior semicircular canal, to the ipsilateral superior oblique and contralateral inferior rectus. When a person's head rotates in a plane perpendicular to the right posterior semicircular canal, for example, movements of the right superior oblique muscle and left inferior rectus muscle (muscles in the same plane of the right posterior semicircular canal) move the eyes in the exact opposite direction, thus stabilizing the retinal image. (Based upon reference 8.)

or even breathing.* The accuracy and efficiency of the vestibuloocular reflex can be easily demonstrated by holding a pencil vertically in front of the face and moving it side to side through a 10-degree arc, 5 times per second. The pencil will appear blurred because the retina cannot compensate quickly enough for the shifting image. If the experiment is repeated with the pencil stationary and the head moved back and forth through the same arc and with the same frequency, the pencil remains sharply defined. The eye movements are *identical* in the two examples, yet only in the second experiment is the vestibuloocular reflex used to keep the pencil in focus.[7]

The vestibuloocular reflex stabilizes retinal images by specific connections between the semicircular canals and eye muscles (Fig. 68.1). When there is unilateral

* A dramatic description of life without the vestibuloocular reflex appears in the story "Living without a balancing mechanism,"[6] written by a physician with bilateral vestibular damage after long-term streptomycin treatment. He describes difficulty reading in bed and having to brace his "head between two metal bars at the head of the bed (to) minimize the effect of the pulse beat, which made the letters on the page jump and blur."

damage to the neural pathways of this reflex, two consequences follow: (1) unopposed stimulation of six eye muscles, three on each side, causes prominent vertigo and nystagmus, and (2) a deficient vestibuloocular reflex is conspicuous when the head is turned to the affected side, a disorder best identified by the head impulse test.

B. HEAD IMPULSE TEST (FIG. 68.2)

First described by Halmagyi in 1988,[10] the head impulse test demonstrates the integrity of the vestibuloocular reflex. The clinician sits in front of the patient and places his or her hands on each side of the patient's head. Throughout the test, the patient is asked to focus on the clinician's nose while the clinician focuses on

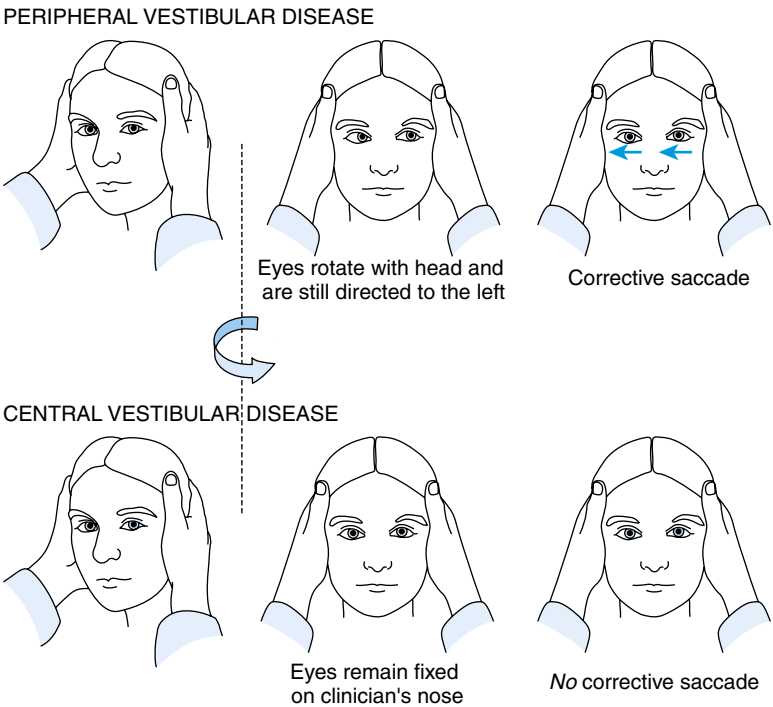

FIG. 68.2 HEAD IMPULSE TEST. The top row depicts the head impulse test in left-sided *peripheral* vestibular disease; the bottom row, in *central* vestibular disease (e.g., stroke). In this example, the clinician is testing the patient's left ear (and left vestibuloocular reflex) by first positioning the patient's head 20 degrees to the *patient's* right (*left* column) and rapidly rotating the head to the straight-ahead position (*middle* column). Throughout the test, the patient is asked to focus on the clinician's nose. The most important observation is what the clinician observes immediately following head rotation (*right* column). In peripheral vestibular disease (*top row*), there is a *corrective saccade* (*arrows*) revealing a deficient vestibuloocular reflex and the patient's attempt to focus again on the clinician's nose. In central vestibular disease (*bottom row*), the intact vestibuloocular reflex allows the patient's eyes to track the clinician's nose throughout the rotation and no corrective saccade appears. When performing the test, neuro-otologists usually start with a warm-up period of slow movements back and forth to help the patient relax, thus permitting the more rapid movements necessary for the test. Most experts perform many trials, randomly to one side or the other; the test is abnormal if the most trials to one side (e.g., two out of three) reveal the corrective saccade. In patients with peripheral disease, the more rapid the initial head movement, the greater the amplitude of the corrective saccade.[9]

the patient's eyes. If the vestibuloocular reflex is intact, the patient can maintain gaze on the clinician's nose during rapid head movements to both sides, and no corrective saccades are observed at the end of the head movement. If the peripheral vestibular system (and vestibuloocular reflex) is abnormal, however, the eyes move away with the rotating head when turned to the abnormal side and, at the end of rotation, the patient's eyes quickly move back to pick up the image of the clinician's nose (i.e., the clinician observes a **corrective saccade**). When compared to asymmetric caloric responses (the traditional definition of unilateral peripheral vestibular disease), the abnormal head impulse test (i.e., corrective saccade present) has a sensitivity of 34% to 57%, specificity of 90% to 99%, positive likelihood ratio (LR) = 6.7, and negative LR = 0.6.[11-13]

In patients with acute vertigo or dizziness, a *normal* vestibuloocular reflex bilaterally (i.e., *no* corrective saccades observed) *decreases* the probability of peripheral vestibular disease and suggests that the cause of the dizziness is central (e.g., stroke).

An excellent online video of the abnormal head impulse test (with corrective saccades) appears in the supplementary material of reference.[14] The only reported complication of the test is complete heart block, observed in a single patient, presumably induced by vasovagal reaction.[15†]

C. SKEW DEVIATION

Skew deviation refers to an acquired hypertropia, which means one eye is aligned higher than the other, a sign of cerebellar or brainstem disease. It is best revealed by the alternate cover test, which is discussed in Chapter 59.

D. ABNORMAL VISUAL TRACKING: SACCADIC PURSUIT

The patient is asked to follow a slowly moving small target (e.g., clinician's finger) horizontally and vertically (the patient's head is still). Most patients have no difficulty following the target (i.e., the pursuit is smooth), but some patients with cerebellar or brainstem disease instead reveal conspicuous quick "catch-up" movements, called **saccadic pursuit**.

E. DIRECTION-CHANGING NYSTAGMUS (FIG. 68.3)

Many patients with acute vertigo have a spontaneous conjugate jerk nystagmus when looking straight ahead. (Chapter 65 defines the terms used to describe nystagmus).[‡] In most patients, whether the disorder is peripheral or central, the nystagmus will persist or worsen when they look *in the direction* of the quick component of the nystagmus. The distinguishing finding appears when the patient looks in the *opposite* direction, which is contralateral to the quick component of the nystagmus. In patients with peripheral disease, the nystagmus diminishes or disappears. In 20% to 56% of patients with stroke, however, it *reverses directions*, a finding called **direction-changing nystagmus**.

A second distinguishing feature of nystagmus is whether the nystagmus is suppressed during retinal fixation (i.e., when the patient is focusing on an object; see Chapter 65). In peripheral disease, the nystagmus diminishes in intensity during retinal fixation; in central disease, it is unchanged.

[†] The authors of this report confirmed the heart block was not due to carotid sinus hypersensitivity.

[‡] In peripheral vestibular disease, the direction of the nystagmus (i.e., its quick component) is *away* from the abnormal side.

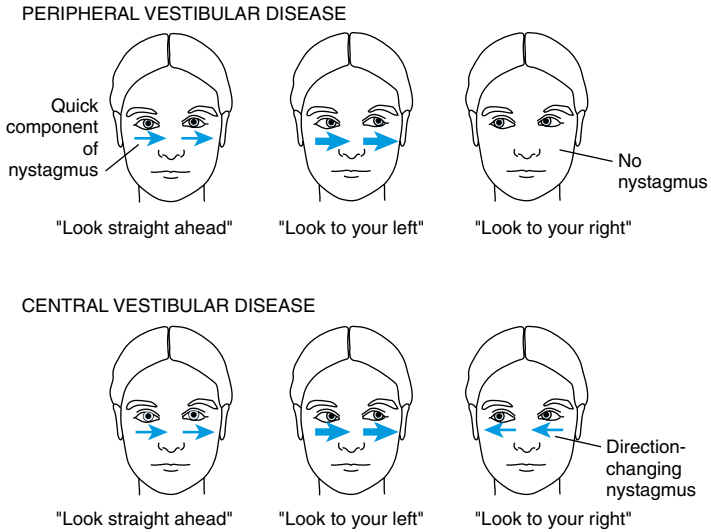

FIG. 68.3 DIRECTION-CHANGING NYSTAGMUS. In this example, the patient has a spontaneous conjugate left-beating jerk nystagmus (*left,* "look straight ahead"; in each example, the arrows indicate the direction of the *quick* component of the nystagmus). The patient is asked to "look to your left" (i.e., the direction of the nystagmus, *middle*) and then "to your right" (the contralateral direction, *right*). In peripheral (*top row)* and central nystagmus (*bottom row*), the nystagmus increases when looking in the direction of the nystagmus ("to your left", *middle*). The distinguishing feature appears when the patient looks in the direction contralateral to the nystagmus ("to your right", *right*). In peripheral disease, nystagmus diminishes or disappears; in central disease, it may *change* directions (direction-changing nystagmus). Importantly, the direction-changing nystagmus must appear before extreme lateral gaze to be regarded as pathologic, because many normal persons have a small amplitude jerk nystagmus on *extreme* lateral gaze.

III. CLINICAL SIGNIFICANCE

Most patients presenting to emergency departments with dizziness, vertigo, or imbalance have benign peripheral disease. Only 3% to 4% ultimately are diagnosed with stroke and most present with obvious focal neurologic findings. For example, diagnostic findings of stroke in patients with dizziness include ophthalmoparesis (LR = 70), visual field cut (LR = 17.5), dysarthria (LR = 10), focal weakness (LR = 9.6), limb ataxia (LR = 9.2), and focal sensory disturbance (LR = 7).[4,5]

In patients with isolated dizziness, additional neuro-ophthalmologic findings help identify patients with strokes.

A. INDIVIDUAL FINDINGS

EBM Box 68.1 presents the accuracy of additional bedside findings in 204 patients with acute vertigo and imbalance, all of whom underwent neuroimaging. The findings that increase the probability of stroke are severe truncal ataxia (unable to sit unassisted, LR = 17.9), *normal* vestibuloocular reflex during the head impulse test (i.e., *no* corrective saccades, LR = 9.6), skew deviation (LR = 5.3), saccadic pursuit (LR = 4.6), and direction-changing nystagmus (LR = 3.5). The presence of smooth pursuit (i.e., absence of saccadic pursuit) and an *abnormal* head impulse test

(i.e., corrective saccade observed, indicating peripheral vestibular disease) decrease the probability of stroke (LR = 0.2).

B. COMBINED FINDINGS

Three oculomotor signs—normal vestibuloocular reflex on head impulse test (i.e., no corrective saccades), direction-changing nystagmus, and skew deviation—are all characteristic of stroke. In studies of acutely dizzy patients, the presence of *any* of these findings increased the probability of stroke (LR = 10.8, see EBM Box 68.1).

EBM BOX 68.1
*Acute Vertigo, Detecting Ischemic Stroke**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio‡ if Finding Is	
			Present	*Absent*
Individual Findings				
Severe truncal ataxia[16]	34	98	**17.9**	0.7
Skew deviation present[16-18]	24-50	86-99	**5.3**	0.7
Saccadic pursuit[17,18]	70-88	80-90	**4.6**	**0.2**
Direction-changing nystagmus[16-18]	20-56	82-98	**3.5**	0.7
Normal head impulse test (i.e., no corrective saccade)[16-18]	60-93	91-98	**9.6**	**0.2**
Combined Findings: (1) normal head impulse test (no corrective saccades); (2) direction-changing nystagmus; and (3) skew deviation[16,18]				
1 or more finding	95-99	86-94	**10.8**	**0.02**

*Diagnostic standard: For *ischemic stroke,* magnetic resonance imaging of cerebellum and brainstem.
†Definition of findings: See text.
‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
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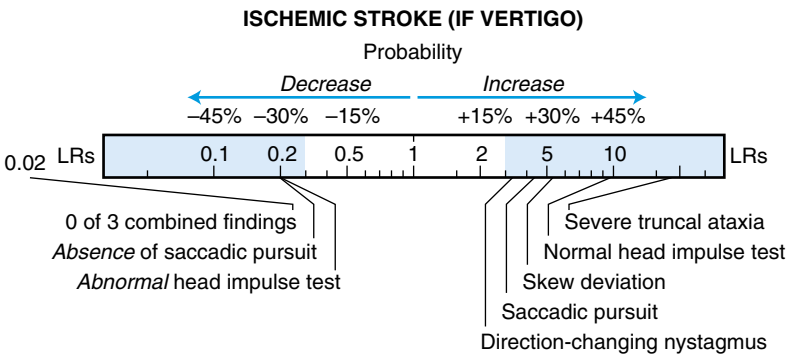

More importantly, the *absence of all three* findings markedly decreased the probability of stroke (LR = 0.02). This LR (0.02) is less than the LR for a *normal* (diffusion-weighted) magnetic resonance image (MRI) (LR = 0.2; i.e., the probability of stroke decreases *more* with the absence of these three findings than it does with a normal MRI result).[§]

The references for this chapter can be found on www.expertconsult.com.

[§] In this study, the diagnostic accuracy of the initial magnetic resonance/diffusion weight imaging for stroke was sensitivity 85%, specificity 98%, positive LR = 44.2, and negative LR = 0.2. The 8 patients with falsely negative MRIs (5 lateral medullary, 1 lateral pontomedullary, and 2 middle cerebellar peduncle infarctions) all had positive repeat MRIs an average of 3 days later.[16]

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